

predominant renal lesion in black patients, whereas immune complex and membranous nephropathy occur more commonly in white patients.¹ Improvements in renal function have been described with highly active antiretroviral therapy (HAART) when the underlying renal lesion is HIVAN or membranous nephropathy.³⁻⁵ We report here an HIV infected patient in whom renal disease caused by hepatitis B induced membranoproliferative glomerulonephritis improved with HAART.

A 34 year old white homosexual man was found to be HIV-1 antibody positive in August 2000 after he presented with biopsy proved Kaposi's sarcoma. At this time he also reported 2 months of fatigue and frothy urine. In the past he had been found to be hepatitis BeAg positive in 1996. Examination revealed multiple cutaneous Kaposi's sarcoma, BP = 170/100, no peripheral oedema, and scanty retinal haemorrhages on funduscopy. Investigations showed blood urea = 9.2 (normal = 2.8–7.6) mmol/l, serum creatinine = 178 (normal = 80–133) µmol/l, normal serum, potassium, and sodium. Liver function tests were normal apart from a serum albumin of 29 (normal = 35–50) g/l. The haemoglobin was 9.3 g/dl and white blood cell and platelet counts were normal. The CD4 count was 110 cells × 10⁶/l and HIV viral load was 47 500 copies/ml. Complement C3 was 0.56 (normal = 0.9–1.8) g/l, C4 was 0.07 (normal = 0.1–0.4) g/l. Immunoglobulin quantification showed normal IgA, IgG = 23.2 (normal = 7.0–16.0) mg/l and IgM = 4.4 (normal = 0.4–2.3) g/l. Hepatitis B serology showed HbeAg+ and HbsAg+ (titre 1:3200). Urinalysis showed blood +++ and ++++ protein. Urine protein = 5.8 g/24 hours and creatinine clearance = 66 ml/min. Ultrasound examination showed normal sized kidneys. Histology of a renal biopsy showed membranoproliferative glomerulonephritis. Staining showed marked deposits of hepatitis B core and surface antigens (fig 1).

The patient was managed conservatively. HAART was commenced with efavirenz, didanosine, and stavudine and hypertension was treated with ramipril. After 8 weeks of HAART the CD4 count was 140 cells × 10⁶/l and viral load was 100 copies/ml. The serum creatinine returned to normal and there was no persistent proteinuria.

This case illustrates the importance of considering non-HIV associated pathology in the HIV infected patient presenting with renal disease. It also shows the value of renal

biopsy in identifying the precise cause of the presentation. This patient demonstrates that non-HIV hepatitis B associated renal disease may improve with HAART. The exact mechanism for this remains unclear.

A SMITH
J D CARTLEDGE
M H GRIFFITHS
R F MILLER

Department of Sexually Transmitted Diseases,
Royal Free and University College Medical School,
Mortimer Market Centre,
London WC1E 6AU, UK

Correspondence to: Dr Miller

rmiller@gum.ac.uk

- 1 Williams DJ, Williams DJ, Williams IG, *et al.* Presentation, pathology and outcome of HIV associated renal disease in a specialist centre for HIV/AIDS. *Sex Transm Inf* 1998;74:179–84.
- 2 Nochy D, Glotz D, Dosquet P, *et al.* Renal disease associated with HIV infection: a multicentre study of 60 patients from Paris hospitals. *Nephrol Dial Transplant* 1993;8:11–19.
- 3 Wali RK, Drachenburg CI, Papadimitrou JC, *et al.* HIV-associated nephropathy and response to highly active antiretroviral therapy. *Lancet* 1998;352:783–4.
- 4 Dellow E, Unwin R, Miller R, *et al.* Protease inhibitor therapy for HIV infection: the effect on HIV-associated nephrotic syndrome. *Nephrol Dial Transplant* 1999;14:744–7.
- 5 Dellow E, Unwin R, Miller R. Presentation, diagnosis and management of renal failure in patients with HIV infection. *AIDS Patient Care STDs* 2000;14:71–2.

Cervical cytology smears in sexually transmitted infection clinics in the United Kingdom

EDITOR,—I found the article by Janet Wilson and Wendy Parsons on behalf of the BCCG, concerning cervical cytology practice in UK genitourinary medicine clinics, comprehensive and reassuring in terms of our practice.¹

The final statement “there is therefore no evidence from this study to support the practice of additional smears in the presence of an effective national cytology screening programme” is both justified and a case well made.

The paper calls additional smears “opportunistic” and recognises them as being performed in women less than the age of 20, women with genital warts, and in some who have had a normal smear within the previous 3 years.

The *OED* definition of opportunity is “favourable, appropriate or advantageous combination of circumstances.”² There is no evidence to suggest that offering smears in these circumstances fulfils this description. If this is so, then we are depriving women such as teenagers of a valuable health screen and patently this is not the case.

I would like to propose, therefore, that we no longer continue to call these smears “opportunistic” but use the term “unnecessary.”

The recognition of this could be an advance for evidence based practice, help to reduce unnecessary anxiety, and release much needed resources.

D A HICKS

Department of Genitourinary Medicine,
Sheffield Teaching Hospitals,
Royal Hallamshire Hospital,
Glossop Road,
Sheffield S10 2JF, UK

- 1 Wilson JD, Parsons W, on behalf of the British Co-operative Clinical Group. Cervical cytology smears in sexually transmitted infection clinics in the United Kingdom. *Sex Transm Inf* 2001;77:107–10.

2 *Collins Oxford English Dictionary*. Ed Patrick Hanks. London and Glasgow: Collins, 1999.

Accepted for publication 22 May 2001

BOOK REVIEW

The Wages of Sin. Sex and Disease, Past and Present. By P L Allen. £17.50; pp 202. Chicago: University of Chicago Press, 2000. ISBN 0 226 01460 6.

This is a profound work describing the impact of venereal diseases and conventional morality in the build up to AIDS. It is written by an American, who has been personally affected by the impact of AIDS. He has written a book on topics in history relating to sex, morality, and infectious diseases, which have had an impact on the public response to AIDS. Throughout, one senses the author's very real loss in what to him and many others have been tragic times.

It is interesting to see how different the general public moral climate is in different societies in the developed world. Thankfully, some forms of evangelism do not have the same influence everywhere.

Does the historical part of the book tell the medical historian anything new? The answer is yes. And that is the gap between what has been known on this subject to academics for a long time and what others are only finding out about now. The chapters containing information on the church's attitude to sexual morality; on leprosy, the early history of syphilis, bubonic plague, and masturbation illustrate the age old story of reactionary view against progress. It is difficult to judge the mores of the past through the views of the present.

It is a pity that the author seems to have given such prominence to those whose views resisted progress. Nothing is mentioned of liberal pioneers in venereal diseases from Van Swieten in the 18th century, through Ricord, Fournier in the next, Abraham Flexner (for the Rockefeller Foundation), Neisser, or indeed the enormous changes brought about by the Royal Commission on Venereal Diseases in Great Britain at the time of the first world war or such notable more recent Americans such as Kampmeier, Stokes, or Earl Moore.

The chapters on America are particularly interesting from a European point of view. Learning about reactionary views always helps in developing any strategy for public knowledge and education. Well educated AIDS lobbyists have certainly had an impact in Europe as in the United States and are neatly described in this work. The bibliography, 14 pages, is particularly good.

This is a book questioning responses and conventional morality in respect, sorrow, and anguish. It is worthy of merit. It enables the modern reader to learn about difficult aspects of morality in relation to venereal diseases and sexuality which have always had more impact on the public than the practising physician.

MICHAEL WAUGH

General Infirmary at Leeds, LS1 3EX

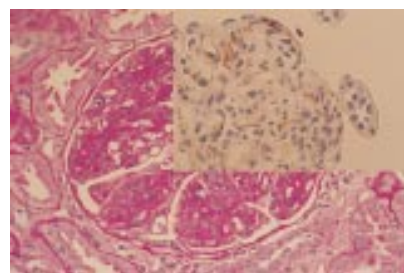


Figure 1 Renal histology shows glomerular mesangial expansion and thickening of the capillary walls, characteristic of membranoproliferative glomerulonephritis. The mesangial areas and capillary walls were positive for IgG, IgA, IgM, and complement components C3 and C1q. There was also positive staining for hepatitis B surface and core (inset) antigens. Haematoxylin and eosin ×400 and immunoperoxidase ×400 (inset).